

**1AN ANALYSIS OF
OBSTRUCTIVE JAUNDICE**

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CERTIFICATE

This is to certify that this dissertation in “AN ANALYSIS OF OBSTRUCTIVE JAUNDICE” is a work done by **DR.J.EMMANUEL THAS**, under my guidance during the period 2006-2008. This has been submitted in partial fulfillment of the award of M.S. Degree in General Surgery (Branch-I) by the Tamilnadu Dr. M.G.R. Medical University, Chennai 600 032.

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INTRODUCTION

Surgical or obstructive jaundice is basically a biochemical derangement resulting in physiological changes due to non deliverance of bile into the intestinal lumen as a consequence of anatomical alterations caused by a variety of pathologies involving the biliary tract, which is mostly incurable with medicines, sometimes palliated by endoscopic procedures and majority of the time cured or palliated by surgery.

Surgical jaundice is a common entity seen in surgical wards or in general practice. The challenge it poses in the diagnosis is totally elusive occasionally, more so in the therapeutic aspect & its outcome.

It is one of the few areas where measures are started, along with diagnostic evaluations, towards preventing and managing the complications.

Even though the advancements in this area have occurred in leaps and bounds, resulting in improved long term results, it still poses to be deceptive in the optimal management with wide variations.

DEFINITION

Jaundice is a generic term for the yellow pigmentation of the skin, mucous membranes, or sclera that is caused by a heterogeneous group of disorders due to hyperbilirubinemia. Although surgical jaundice is characterised by hyperbilirubinemia and dilated bile duct it is clearly inadequate to equate the two. Similarly obstruction and the dilation of the common bile duct are not synonymous, though they complement each other.

Complete biliary obstruction produces jaundice.

Incomplete obstruction produces symptoms and biochemical changes but may or may not be associated with features of clinical jaundice.

Chronic incomplete obstruction with or without clinical symptoms or biochemical features, produces pathological changes in bile ducts or liver.

Segmental obstruction involves one or more isolated segments of the biliary tree which may take the form of complete, intermittent or chronic incomplete obstruction.

AIM OF THE STUDY

To study the incidence, etiopathogenesis and progression of disease in obstructive jaundice.

To investigate and analyse all patients with obstructive jaundice and to prepare them for surgical intervention.

To plan for surgical intervention, either curative or palliative.

To follow up the patients to understand the progression of the disease, after surgical intervention and to analyse the outcome.

MATERIALS AND METHODS

Obstructive jaundice patients admitted in all the four surgical units of Govt. Royapettah Hospital, Kilpauk Medical College, Chennai. between May 2006 to October 2008 were studied and evaluated.

On admission a detailed history and clinical assessment of the problems were made. Preliminary biochemical investigations were carried in all the patients followed by real-time ultrasonography and ERCP if necessary. In all patients CT scan of abdomen was carried out to assess the operability.

If operable lesion were detected patients underwent a careful preoperative preparation. Histopathological examination was conducted in relevant patients. They were followed in the post operative period and subsequent to their discharge.

The various causes for the obstructive jaundice in our hospital was evaluated. A comparison was made with other studies regarding the incidence, mode of presentation, prognosis and survival. The results were compared and graphically represented and a conclusion was arrived from it.

ANATOMY OF HEPATOBILIARY TRACT

ANATOMY OF THE HEPATOBILIARY SYSTEM

Liver is the largest gland in the body weighing about 1500 gms and receiving 1500 ml. of blood per min. It arises from the foregut endoderm. It has three surfaces, two lobes and eight segments, four for each side, each side being separated by Cantlie's line or main portal scissura, thus leaving a smaller right liver than the right lobe.

It has the following ligamentous attachments;

Falciform ligament

Right and left triangular ligaments

Coronary ligaments

Gastrohepatic omentum

The biliary tract begins as blind channels or canaliculi, that ramify between the hepatocytes, formed by the specialised structures in the cell membranes of adjacent hepatocytes, and drain into the interlobular bile duct in the portal triad which forms the periphery of the functional unit called lobule, centered around central vein.

The right hepatic duct is formed by the intrahepatic union of dorsocaudal and ventrocranial branches (segments V-VIII) and the left duct by the medial and lateral branches.

EXTRAHEPATIC BILIARY APPARATUS :

Consists of

Hepatic ducts - right, left and common

Gall bladder

Cystic duct

Common bile duct

Gall Bladder

It is a pear shaped reservoir of 10 cm length with capacity of 50 ml. Situated in the gall bladder fossa in the inferior surface of liver. It is covered with peritoneum variably.

It has three parts :

Fundus - has the poorest blood supply;

Body

Neck or infundibulum - frequently has the abnormal dilatation called Hartmann's pouch in the posteromedial wall, which may adhere to the surrounding structures obscuring the anatomy of the region.

Cystic duct :

Variable course of 3-4 cm. Usually, downward and backward, joining the common hepatic duct, guarded by a false valve formed by the mucous fold called valve of Heister.

Hepatic ducts :

Right and left unite to form common duct (usually extrahepatically 90% within 1 cm.) which is about 3 cm. At the porta, they are arranged behind forwards as portal vein, hepatic artery and hepatic ducts.

Common bile duct:

Anatomically starts distal to cystic duct junction but surgically from the union of right and left hepatic ducts. It measures about 10-12 cm. In length with average diameter of 7 mm. (Range 4-10mm). It is divisible into supraduodenal, retroduodenal, intrapancreatic and intraduodenal. It unites with the main pancreatic duct to form the ampulla and opens into the major papilla in the posteromedial wall of II part of duodenum.

SURGICALLY RELEVANT VARIATIONS IN ANATOMY

The knowledge of segmental anatomy is deployed in the hepatic resections. The hepatic arterial, portal venous and hepatic bile duct branches conform to

the segment organisation excepting the hepatic vein.

The Rex recessus in the umbilical fissure in the anterosuperior surface, is followed by the ligamentum teres to access the segment III and the left duct in the round ligament approach for bypass in inoperable cholangio-carcinoma.

A thorough knowledge of not just the anatomy but also the variations is needed to avoid complications. In Gall Bladder, commonest anomaly is the “Phrygian cap”. Others include agenesis, floating GB, sausage shaped GB, positional anomalies, trabeculated GB.

Bile duct anomalies include

- aberrant, supernumerary or accessory ducts.
- variations in the confluence of major intrahepatic ducts.
- variations in the course and termination of cystic duct.
- variations in the union with pancreatic duct at the termination of CBD.

Vascular anomalies :

Of importance is the cystic artery, which is normally a branch of right hepatic artery, emerging from behind the common hepatic duct.

- Accessory cystic artery
- Low origin of the cystic artery.
- Abnormal origin from other vessels
- Short cystic artery with a looped right hepatic artery called

The Moynihan's hump.

- Close lie of the right hepatic artery to the cystic duct with a

Short cystic artery - most dangerous anomaly.

The relevance of peri choledochal arterial plexus, formed by the duodenal branch of gastroduodenal and right hepatic arteries, in the prevention of strictures should be understood.

AETIOLOGY OF BILIARY OBSTRUCTION

AETIOLOGY OF BILIARY OBSTRUCTION

It is important to recognise the various causes of biliary obstruction, since they produce many subtle clinical syndrome whose true nature may go unrecognised, the clinician should recognise at least 4 categories of biliary tract obstruction discussed before :-

- Complete biliary tract obstruction
- Intermittent obstruction
- Chronic incomplete obstruction

- Segmental obstruction

A list of lesions commonly associated with biliary obstruction is given in Table-I.

Management wise it is easier to conceptualize the causes as those affecting extra hepatic obstruction or intra hepatic mechanical obstruction.

TABLE - I

COMPLETE OBSTRUCTION

- Tumours, especially of pancreatic head
- Ligation of common bile duct
- Cholangiocarcinoma
- Parenchymal liver tumours

INTERMITTENT OBSTRUCTION

- Choledocholithiasis
- Periapillary tumours

- Duodenal diverticula
- Papillomas of the bile duct
- Choledochal cyst
- Poly cystic liver disease
- Intra biliary pressure
- Haemobilia

CHRONIC INCOMPLETE OBSTRUCTION

- Stricture of the common bile duct
- Stenosed biliary - enteric anastomosis
- Stenosis of Sphincter of Oddi
- Chronic pancreatitis
- Cystic fibrosis

- ? Dyskinesia

SEGMENTAL OBSTRUCTION

- Traumatic (including iatrogenic)
- Hepatodocholithiasis
- sclerosing cholangitis
- cholangiocarcinoma

EXTRA HEPATIC BILIARY OBSTRUCTION

The most common obstacle to the flow of bile are stones, tumours and strictures. Stones in the common bile duct may lead to sufficient dilatation of the proximal ducts that bile may flow around them, thus minimising hyperbilirubinemia. Stones in the cystic duct may impact on the common duct (Mirrizzi syndrome).

Benign and malignant intrinsic tumours of the Bile ducts result in Jaundice, as do extrinsic tumours of the bile ducts that directly invade the duct wall and fix it.

Metastatic tumours in the porta hepatic lymph nodes ordinarily do not cause jaundice as they tend to push the duct aside rather than fix it. The most frequent cause is carcinoma of the colon. Occasionally, histiocytic non-Hodgkin's lymphoma, Hodgkin's disease, or even chloroma may extend from lymph nodes of the retroperitoneal area into the walls of the ducts and cause jaundice. Metastatic involvement of hilar connective tissue of liver rarely produces obstructive jaundice.

Strictures of the bile duct, both following surgical trauma of bile ducts and primary sclerosing cholangitis usually lead to incomplete but sometimes infected obstruction. Rare causes include parasitic and mycotic conditions, choledochal cysts, duodenal diverticula or hepatic artery aneurysms. The role of "inspissated bile syndrome" is not established.

INTRAHEPATIC MECHANICAL BILIARY OBSTRUCTION

This is per se seldom extensive enough to produce jaundice. Carcinoma at the bifurcation of the common bile duct beginning in one hepatic duct and may involve the other and cause cholestasis. Suppurative cholangitis was previously a common cause of jaundice. Various forms of biliary dysplasia, such as congenital hepatic fibrosis, cystic disease of the liver or Caroli's disease may be complicated by secondary bacterial

infection of the bile ducts. Even solitary non parasitic cysts may obstruct the biliary tree.

Chronic pancreatitis is recognised as a cause of cholestasis, mainly as a result of cholangiographic or ultrasonographic demonstration of formidable narrowing of adjacent bile duct. The protracted jaundice usually results from large amounts of alcohol damaging the liver, since despite the chronic pancreatitis, the bile duct is almost normal. Hydatid cysts can obstruct ducts.

In cystic fibrosis of the pancreas, inspissated mucus in ductules and ducts may cause partial obstruction without jaundice. Metastatic carcinoma is frequently associated with cholestasis, so are lymphomas and Hodgkin's disease.

Cholestatic jaundice is a serious problem after transplantation of the liver. In the immediate postoperative period, it may be caused by complications mechanical or infectious, at the biliary anastomosis. A few weeks postoperatively, rejection phenomenon may be the reason. Eventually bile ducts may disappear and the lesion may resemble primary biliary cirrhosis.

PATHOPHYSIOLOGY OF BILIARY TRACT OBSTRUCTION

Biliary obstruction results in many physiological, pathological, biochemical and functional changes which are considered in detail below.

PHYSICAL EFFECTS

The normal secretory pressure of bile is 120-250 mm. H₂O (11-8-24.5 kPa). When the intra biliary pressure is raised to more than 300 mm. H₂O (29.4 kPa), there is total inhibition of bile secretion. However the effects on secretion of bile salts, cholesterol and phospholipids are unequal and the return of the secretory functions following relief of the bile duct obstruction may be asynchronous. The degree of proximal biliary dilatation depends on nature and duration of obstruction and also on pliability of the extra hepatic bile ducts and of supporting skeleton of intra hepatic bile ducts. The former may be restricted in inflammation and fibrosis; the latter may be enhanced by impaired hepato cellular function raising suspicion of portal hypertension. These two pathologies can cause a lack of proximal biliary dilatation inspite of organic distal lesion.

EFFECT ON BLOOD FLOW

Though presumably obstruction of bile ducts should increase intrahepatic hydrostatic pressure and produce an elevation of hepatic sinusoidal pressure, thus adversely affecting hepatic tissue perfusion; this however has been disproved in animal experimental studies. Chronic obstruction, however has been demonstrated to lead to fibrosis with secondary portal hypertension.

PATHOLOGICAL EFFECTS

DUCTULAR CHANGES

The initial changes occur at the canalicular level and are mediated by high local concentration of bile salts. There is dilatation of centrilobular bile canaliculus, bile thrombi are present in the lumen. In more prolonged cholestasis the bile ducts and ductules appear to be increased probably both by lengthening and tortuosity and by sprouting of small bile ductules secondary to inflammation; these changes are probably due to lithocholate or altered portal micro circulation.

FIBROTIC CHANGES

Intra hepatic fibrosis occurs secondary to cholangitis which causes laying down of

reticulin fibres which is followed by formation of hard collagen. The changes in extrahepatic ducts are a sequence of mucosal atrophy and squamous metaplasia followed by inflammation and ultimately fibrosis in the subepithelial layers of the ducts.

HEPATOCTYTE CHANGES

Cholestasis causes local high concentration of bile salts which inhibit the enzyme cytochrome p-450 causing damage along the biliary pole of the Hepatocyte. This initially results in “feathery necrosis” and in its most severe form is associated with leukocyte infiltration resulting in “Biliary piecemeal necrosis”.

BIOCHEMICAL EFFECTS

BILIRUBIN

The rise in serum bilirubin varies in intensity with the type of obstruction - whether complete / intermittent / chronic complete / segmental. Persistent absence of urobilinogen from urine is strong evidence of obstructive jaundice and bilirubin is usually present in the urine until the secondary hepatocellular disease becomes advanced. It has been proposed that it is bile concentration gradient that opposes the

transport of bilirubin into the canaliculus, and this gradient may be produced by stasis in the biliary tree. The mechanism by which conjugated bilirubin reflexes into sinusoidal plasma in cholestasis is unclear.

BILE ACIDS

Complete biliary tract obstruction will interrupt the enterohepatic circulation of bile salts. Serum bile acids may be very high ranging from 4-60 times the normal . Nevertheless the synthesis itself is impaired. There is an increased urinary excretion of bile acids but this still amounts to a very small proportion of faecal excretion. In addition abnormal bile acids are produced by the liver, including ursodeoxycholate, which are more easily Excreted in urine than the normal bile acids. The pruritus of cholestasis is probably caused by deposition of bile acids in the skin. External biliary drainage of the obstructed tract results in depletion of the bile salt pool and its replacement may have a beneficial effect on the clearance of bilirubin and other moities excreted by mechanisms dependent on bile salt related canalicular flow.

ALKALINE PHOSPHATASE

This is a sensitive marker of biliary obstruction but its lack of specificity is a clear pitfall. In the liver, the isoenzymes of alkaline phosphatase exist in the plasma membrane of the microvilli of the canaliculus. Sources of other isoenzymes are bone, intestine, kidney and placenta. The return to normal of alkaline phosphatase after relief of biliary obstruction may be a good index of successful therapy in majority of cases. A simple practical application of the measurement of alkaline phosphatase in the complex post operative patient, particularly following biliary reconstruction or major hepatic trauma elevation of serum bilirubin in the absence of raising alkaline phosphatase may indicate an intraperitoneal biliary collection while parallel increases of bilirubin and alkaline phosphatase suggest a degree of obstruction.

OTHER BIOCHEMICAL PARAMETERS

SERUM AMINO TRANSFERASES

Gross elevation of aminotransferase is an index of hepatocellular damage. Minor elevation is common, however, in extrahepatic biliary obstruction, and may even rise earlier than alkaline phosphatase in case of acute obstruction. The levels fall to normal nearly so, within several days, despite depending jaundice. However in later stage they

may rise again, and this is an adverse prognostic feature, indicating prolonged obstruction with superadded intrabiliary infection or invasive sepsis from suppurative cholangitis. Gamma Glutamyl transpeptidase tends to parallel alkaline phosphatase and is sometimes used as an alternative to the isoenzyme fractionation of alkaline phosphatase.

SERUM PROTEINS

Depression of the serum albumin is a common feature in patients with malignant biliary obstruction, particularly in prolonged nutritional impairment. Elevation of globulins particularly the IgG fraction, may be seen in severe parenchymal liver disease due to long standing obstruction. Gross elevation of gamma globulins may be related to porta-systemic shunting and should raise a strong suspicion of secondary portal hypertension.

LIPID METABOLISM

This is grossly altered but most of the changes are unhelpful because of lack of specificity. Cholesterol may be elevated in long standing biliary obstruction.

FUNCTIONAL EFFECTS

The fundamental dearrangements due to cholestasis are reflected in the hepatocellular function and reticulo endothelial function.

HEPATOCELLULAR FUNCTION

The alterations in hepatocellular function occur due to

1. Retained bile salts – which have a toxic effect on the cellular

Component cytochrome P-450 and transform it into inactive

Cytochrome P-420.

2. Retained bilirubin – which inhibits mono oxygenease activity
3. Other moities - disruption of mitochondrial functions.

Though various enzymes which are markers of hepatocellular dysfunction are routinely used, a dynamic indicator of liver function would be a valuable adjunct to assessment of patients who are candidates for major surgery. The clearance of antipyrine (a minor analgesic almost entirely oxidized in the liver) and paracetamol (which is conjugated to glucoronide and sulphate esters in the liver) are effective parameters of

liver function. Abnormal glucose tolerance is also seen and may be due to defects in the hepatic alkaline cycle.

RETICULO ENDOTHELIAL FUNCTION

Kupffer cells lining the sinusoids have a major role in the inactivation of endotoxins, the abnormal lipopolysaccharides derived from cell wall of gram negative bacteria in the gut. In biliary obstruction this function is impaired and systemic endotoxemia occurs more frequently in jaundiced patients in comparison to non jaundiced patients. This fact has been demonstrated in animal experiments. Endotoxemia is associated with postoperative renal failure in the jaundice patient; hence these results are of therapeutic significance.

SECONDARY EFFECTS

INFECTION :

The normal biliary tract is sterile. Following surgical or radiological intervention of the obstructed biliary tract the bile culture rate increases. Therefore fluid suppurative cholangitis is commoner in malignant obstruction rather than benign biliary obstruction. The most common infecting organisms are *Escherichia coli* and *Streptococcus faecalis*, this spectrum may change following radiological intubation or surgery.

ATROPHY

The liver atrophy may be uniform, lobar or segmental. Uniform atrophy occurs in presence of malnutrition or partial occlusion of the main stem of portal vein.

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Segmental or lobal atrophy occurs due to portal venous occlusion / bile duct occlusion / proximity to space occupying lesion / (rarely) unilobular veno-occlusive disease. Atrophy can be determined radiologically by isotope or CT scans or transhepatic cholangiograms.

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CLINICAL PRESENTATION

CLINICAL PRESENTATION

The presenting feature may be in many ways in any combination of the following :

JAUNDICE

A generic word from French meaning yellow. It is due to the deposition of bile pigments in sclera, skin, and mucous membranes. The predilection of sclera is due to the high affinity of the elastin, to bilirubin, in the sclera.

WEIGHT LOSS & ANOREXIA

Presence of these two factors are more commonly indicative of malignancy where the history is usually progressive.

PAIN

The site and type of pain vary with the underlying pathology e.g., Pain in epigastrium radiating to back indicates a pancreatic pathology, where pain is usually due

to involvement of retropancreatic nerves and pancreatic duct obstruction.

PRURITIS

It strongly suggests extrahepatic obstructive jaundice, even in infective pathology it indicates presence of cholestatic component. It may begin abruptly and is worse in the nights but not usually relieved by ointments, lotions etc.

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FEVER

Fever and rigors indicate cholangitis and high degree of suspicion is needed as it needs urgent attention. Care should be taken to rule out other causes of fever.

DYSPEPSIA

Quite commonly seen in gallstones esp. to fatty foods, but cannot attribute to any other disorders, specifically. Bloating may be seen in obstruction of duodenum.

HIGH COLOURED URINE

Due to bilirubinuria. It may be noted several days before the onset of jaundice.

The darkest specimen is the first passed urine in the morning.

PALE STOOLS

Absence of the bilirubin metabolites lead to clay coloured to pale yellow stools, the colour varying with the degree of cholestasis.

MALaise AND FATIGABILITY

May be due to poor nutrition or due to underlying psychiatric disorder eg.

Depression in pancreatic carcinoma which has to be differentiated.

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PHYSICAL SIGNS

JAUNDICE

The depth of jaundice depends on the type and duration of obstruction e.g. Progressive and deep jaundice in carcinoma head of pancreas and intermittent in periampullary carcinoma and choledocholithiasis.

DISTENDED GALL BLADDER

Palpable gall bladder in the presence of jaundice goes more in favour of obstruction caused by a malignancy. Courvoisier's law reinforces this.

PALPABLE LIVER

Liver is usually enlarged in cases of pure extrahepatic obstruction, absence of which should prompt the clinician to look for other pathologies. Nodularities over the liver suggest malignant involvement, esp if obstructive type of jaundice, and point to a extrahepatic cause.

MASS

Both benign and malignant causes can present with mass abdomen, viz choledochal cyst and carcinoma head of pancreas respectively. The character of the

mass indicates the type of lesion.

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ASCITES

This usually signifies extensive spread of a malignant disease, however this can happen in the absence of peritoneal metastasis too, like due to portal vein obstruction at the porta hepatis.

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INVESTIGATIONS

INVESTIGATIONS

It is to reiterate that history and examination alone can give a diagnosis in 80% of the patients.

BIOCHEMICAL

BILIRUBIN

Their intensity differs with the type of obstruction. Persistence or absence of urobilinogen in urine is a strong evidence of obstructive jaundice.

Associated secondary hepatocellular disease can produce bilirubin in urine.

Clinically jaundice is apparent when the level of bilirubin is above 2mg% or 3mg% when the jaundice is rising rapidly. Also in convalescence it is seen until the level falls to 1.5 mg% as it still remains bound to sclera.

Bilirubin in urine is a sensitive screening test. Yellow foam on shaking the urine and its reaction with diazotized 2,4 dichloroaniline or a stable diazonium salt in the presence of acid buffer produce a colour. This is the basis of van den bergh reaction.

Urine urobilinogen is characteristically absent in obstructive jaundice as entry into bowel is needed for its production, and subsequent absorption to appear in urine.

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I

The van den Bergh reaction differentiates the type of jaundice. A direct reaction indicates immediate colour change (in cholestasis) while pre-treatment with alcohol or urea is needed to produce a colour change in haemolytic jaundice.

BILE ACIDS

Obstruction of bile flow can cause an increase in the bile acid level by 4-60 fold but at the same time impairs the synthesis. But the abnormal bile acid secretion increases which is more avidly excreted in urine along with normal acids too.

ALKALINE PHOSPHATASE

It is sensitive but lacks specificity in obstructive jaundice. This is elaborated also

by bone, leucocytes, kidney, reticulo-endothelial cells, intestine, placenta and tumours, which can be differentiated by electrophoresis. It is a plasma membrane enzyme that rises with jaundice to reach a plateau 2-4 times normal. But it decreases slowly when jaundice fades. This is a good indicator of the adequacy of treatment.

Post operatively, in patients following hepatobiliary surgery, a rising bilirubin with this enzyme indicates obstruction or if the bilirubin alone increases it indicates collection intra-abdominally.

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SERUM AMINOTRANSFERASES

Serum aspartate aminotransferase (AST : Syn : SGOT- sr glutamic oxaloacetic transaminase) and serum alanine amino transferase (ALT; syn SGPT - sr glutamic pyruvic transaminase) are indicators of hepatic damage. They may rise earlier than alkaline phosphatase in acute obstruction but return to normalcy even if obstruction persists. ALT is more specific than AST.

SERUM PROTEINS

Decrease in albumin levels is seen in malignancy, with poor nutrition also contributing to it. While elevations of globulins esp gamma IgG fraction is seen in parenchymal liver disease and in porta-systemic shunting and should raise the suspicion of secondary portal hypertension.

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IMAGING STUDIES

It ranges from simple X-ray to sophisticated studies like magnetic resonance imaging and radionucleotide studies.

PLAIN X-RAY ABDOMEN

Can diagnose

- Calculous in the biliary tree
- Gallstone ileus and gas in biliary tree
- Calcification elsewhere like in pancreas and in gall bladder wall

(Best detected by plain X-ray) or milk of calcium bile.

- Acute emphysematous cholecystitis.

BARIUM MEAL SERIES

- Widening of C-loop, PAD sign in carcinoma head of pancreas
- reversed 3 sign-in periampullary carcinoma
- smooth filling defects in the ampulla due to stone
- irregular filling defect in duodenal growth

INTRAVENOUS CHOLANGIOGRAPHY

It uses sodium iodopamide (biligraphin) which has more concentration of iodine.

About 20 ml. in infusion form is given. Iotroxate meglumine is also used.

It is superior in visualising the biliary system but inferior for GB. It is an accurate technique for diagnosis of cystic duct obstruction.

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REALTIME ULTRASONOGRAPHY

It is the first line investigation for biliary tract and pancreatic diseases and in cholestatic jaundice.

ADVANTAGES

- It detects gallstones and gallbladder diseases.
- It detects lesions of biliary tract and hepatic parenchymal diseases.
- It detects lesions in pancreas
- accuracy of diagnosis of extrahepatic obstruction is >90%

DISADVANTAGES

- Inferior to oral cholecystography for adenomyomatosis of

Gall bladder
- Cannot give the exact site and extent of the lesion causing

Cholestasis.
- It is unsatisfactory in the following
 - obese
 - ascites
 - following previous surgery
 - gaseous distension of upper abdominal viscera

By convention the diameters of the CBD is measured just above the junction of the cystic duct. A CBD whose diameter exceeding 10mm. is considered dilated. In the absence of contrast e.g. USG or MRCP, diameter above 7mm indicates dilatation.

COMPUTED TOMOGRAPHY

Reserved for patients for whom USG has failed.

ADVANTAGES

- Contrast enhancement increases diagnostic yield.
- High dose contrast helical CT has better detection of solid

lesions of extrahepatic bile ducts, liver and pancreas.

DISADVANTAGES

- Cost and radiation exposure

PERCUTANEOUS TRANSHEPATIC CHOLANGIOGRAPHY

Used for visualization of the biliary tract in the jaundiced patient and can be modified to allow percutaneous transhepatic drainage and insertion of prostheses, where the drainage is only attempted palliatively and not as preoperative procedure as

decompression offers no advantage. Success rates in dilated duct system is approx 100%

It is done with 22 G Chiba needle under sedation with fluoroscopic control.

Systemic antibiotics are needed. It is used when ERCP fails or does not provide information on the proximal duct system due to obstruction.

COMPLICATIONS

- Septicaemia
- Bile leak
- Haemorrhage and hemobilia
- Pneumothorax
- Intrahepatic arterio-portal fistula
- Bile embolisation

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ENDOSCOPIC RETROGRADE CHOLANGIOPANCREATICOGRAPHY

It provides useful information in cholestatic jaundice irrespective of the dilatation of biliary tree. It is done through a side viewing endoscope. It is both diagnostic and

therapeutic (stone removal, stenting, sphincterotomy)

It is very accurate in diagnosing

- tumours of the bile ducts and pancreas
- sclerosing cholangitis
- ductal calculi

DISADVANTAGES

- needs technical expertise,
- complications (haemorrhage, pancreatitis, cholangitis, duodenal Perforation, cholecystitis, gallstone ileus).

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MAGNETIC RESONANCE CHOLANGIOPANCREATOGRAPHY

PRINCIPLE : The static bile ensures very high signal in T2 weighted images, in

contrast to the surrounding liver with blood supply and hence produces no signal.

It is very useful in

- Biliary calculi - detects as small as 2 mm. stone. It is of choice in the

Diagnosis of intrahepatic stones.

- Choledochal cyst - it displays all types of cysts
- Bileduct injury - outlines both duct and collection. So ERCP needed.
- Bileduct tumours - esp. High bile duct tumours, detects extension

Of tumours along intrahepatic ducts, i.e. Complete staging, and

Assesses patency of hepaticojejunostomy after surgery.

- Pancreatic disease - highly accurate for pancreatic divisum, and in

Ampullary and cystic tumours.

MRCP > ERCP

- for duct stones in gall stone

Pancreatitis

- high bile duct tumours
- to differentiate the cause of common

Hepatic duct obstruction viz GB ca,

ERCP > MRCP

- minimal disease chronic

pancreatitis

- when intervention is planned
- to check whether bile duct

injury is active or not

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BILIARY SCINTISCANNING

Compounds used are ^{99m}Tc -labelled iminodiacetic acid (IDA) and diethylacetanilido - iminodiacetic acid (EHIDA). They are selectively taken up by hepatocytes and secreted in bile, which is imaged by a gamma camera. This is not influenced by the presence of cholestasis.

- It is the most accurate test for acute cholecystitis irrespective of the nature.

- Normal gall bladder scan virtually rules out (100%) cholecystitis while false positive study is not uncommon.
- Only indication for its routine use in jaundiced patient is in Neonates for biliary atresia.
- It can however differentiate the various types of jaundice. Viz. Complete or partial obstruction and hepatocellular varieties.
- Functional evaluation of bilio-enteric anastomoses.
- To check bile leaks.

INTRAOPERATIVE FLOUROCHOLANGIOGRAPHY

It is commonly performed through cystic duct and is now considered a part

Of cholecystectomy.

It provides the best mapping of the biliary tree. The commonest artefact is air bubble.

Criteria for normal cholangiogram.

- unequivocal flow into duodenum
- both intra and extra hepatic duct should be visualized
- no filling defects
- main portion of the duct is of normal calibre
- narrow terminal portion
- no excess retrograde filling of extrahepatic duct.

T-TUBE CHOLANGIOGRAPHY

It is usually performed after CBD exploration 7-10 days with a water soluble contrast. It has been replaced by completion choledochoscopy.

Before injecting contrast, T-tube and the extrahepatic bile duct is filled with 60 ml. Of saline to remove air bubbles.

CHOLEDOCHOSCOPY

It is now an integral part of common bile duct exploration. Both rigid and flexible scopes are used.

ADVANTAGES :

- Better evaluation of intradochal pathology
- Allows biopsy and removal of impacted stones
- Can be passed via T-tube in post op cases replacing

Cholangiography

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Gallstones

DISADVANTAGES

- needs expertise and can be traumatic.

LAPAROSCOPY

Demands to have its routine use in all patients with jaundice of malignant etiology. It allows visualization of all the organs and biopsy and cytology can be

obtained. It can preclude unnecessary laparotomies in advanced cases.

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PREOPERATIVE PREPARATION

All the patients are placed under the category of urgent cases. Adequate timing of intervention and preparation of the patient are essential as delays more than 4 weeks increase morbidity and mortality.

PREVENTION OF INFECTIVE COMPLICATIONS

The normal biliary tract and the human bile are sterile and obstruction predisposes to infection more with stones than with malignant cause. Aerobic organisms are more common and most commonly due to gram negative bacilli. Post op infection in the form of cholangitis and septicemia occurs with infected bile and needs prophylaxis, which in general is given for high risk cases. They are :

- all jaundiced patients
- patients with rigors and pyrexia
- elderly
- undergoing emergency biliary procedures
- CBD stones if not jaundiced
- secondary biliary intervention

CORRECTING COAGULATION DISORDERS

The most common disorder of coagulation is prolonged prothrombin time due to deficiency of Vit-K dependent factors. The intramuscular injection of phytomenadione 10-20 mg. Will reverse it in 1-3 days. FFP can be used perioperatively where the

reason can also be due to DIC due to endotoxemia.

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PREVENTION OF RENAL FAILURE

The underlying mechanism is probably reduced glomerular filtration or endotoxemia. Good hydration and pre op natriuresis reduces the incidence. Both osmotic and loop diuretics are effective to maintain an output of above 40 ml./hr. Pre op oral chenodeoxycholate and lactulose, act by decreasing the absorption of endotoxin, give variable results.

FLUID AND ELECTROLYTE CORRECTION

Hypokalemia is the frequent abnormality and in general isotonic saline use should be restricted as the exchangeable body sodium is increased. Oral diet is the safest and used whenever possible. Parenteral nutrition should be restricted to grossly malnourished patients coz of infective risk. High intake of carbohydrates and low aromatic aminoacids are advised.

PREVENTION OF HEPATIC ENCEPHALOPATHY

It is common in patients with prolonged complete large bile duct obstruction or with chronic hepatocellular disease. If jaundice is above 15gm% or if the patient shows signs of impending failure a period of decompression is needed, in the form of sphincterotomy for periampullary growth or stenting for duct obstruction. Correction of hypokalemia restriction of sedatives, hypnotics, analgesics and infections are crucial.

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OPERATIVE PROCEDURES

OPERATIVE PROCEDURES

CHOLEDOCHOLITHIASIS WITH CHOLELITHIASIS

There are basically two options.

- (1) ERCP - Sphincterotomy or papillotomy with stone retrieval

With CBD stenting followed by open / laparoscopic cholecystectomy.

- (2) Open / laparoscopic cholecystectomy with CBD exploration with

T-tube drainage.

CARCINOMA HEAD OF PANCREAS

Curative surgery

There are four options :

1. Whipple operation (pancreaticoduodenectomy)

Removal of head and neck of pancreas with duodenum, distal half of

stomach, lower common bile duct, gall bladder and upper jejunum with as much regional nodes as possible.

2. Pylorus preserving Pancreaticoduodenectomy

Introduced to modify the post gastrectomy symptoms associated with antrectomy. It differs technically from Whipple's by preservation of blood supply to proximal duodenum.

3. Total Pancreatectomy (Total Pancreaticoduodenectomy)

Along with Whipple's excision of spleen, body and tail of pancreas, thorough regional lymphadenectomy.

4. Regional Pancreatectomy

Entails extirpation of transpancreatic portion of portal vein, celiac axis, superior mesenteric artery and middle colic vessels with total pancreatectomy.

PERIAMPULLARY CARCINOMA

1. Pancreaticodenectomy

This is the procedure of choice and done when ever the patient is fit and the lesion operable.

2. Local excision ;

Used for malignant lesions if

A) the patient is unfit for major surgery

B) tumour limited to ampulla of vater with favourable grading

And no infiltration.

3. Transduodenal excision.

Only if the lesion is benign as diagnosed by preoperative biopsy or papillectomy endoscopically.

CHOLANGIOCARCINOMA

Treatment depends on the site of the growth

1. Middle third :

Excised from just below the confluence down to duodenum with

pancreaticoduodenectomy in continuity with associated hepatoduodenal and retroduodenal nodes.

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2. Distal third

They follow the treatment of periampullary carcinoma viz.

Pancreaticoduodenectomy

3. Hilar lesion

If along the right or left ducts, it needs resection including lobes. If it is beyond the junction of two ducts reconstruction with hepaticojejunostomy is planned.

CARCINOMA GALL BLADDER

Surgical options depend on the staging

1. Extended cholecystectomy - For stage I-III i.e., Upto serosal involvement, cholecystectomy with 3 cm. Of hepatic parenchyma with lymph node clearance.

2. Even more aggressive approach of right lobectomy with extensive lymphadenectomy is done for advanced disease.

PALLIATIVE OPERATIONS

They are done for incurable disease not with the intent to improve survival but to better the quality of life.

The options are

1. Cholecystojejunostomy
2. Choledochoduodenostomy
3. Choledochogastrostomy
4. Choledochojejunostomy
5. Cholecystoduodenostomy

The choice of procedure depends on the type of the disease.

Of common use in cholecystojejunostomy with Roux-en-Y loop or jejunal loop. Gastrojejunostomy is performed where there is a probability of gastric outlet obstruction.

CHOLEDOCHAL CYST

The procedure of choice is surgical excision with Roux-en-Y anastomosis. Hepaticojejunostomy, choledochoduodenostomy, choledochojejunostomy, are some of the procedures used.

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RESULTS AND OBSERVATIONS

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RESULTS AND OBSERVATIONS

From May 2006 to October 2008 we had 57 patients of biliary obstruction in our

wards in Government Royapettah Hospital, Chennai. Of the 57 patients , 34 (59.65%) were male and 23 (40.35%) were female.

The mean age group of patient was 49.16 years. The range of age being 19 years to 76 years.

The mean duration of illness was 5.4 months, the range being 7 days to 18 months 53 pts. (92%) had jaundice, 32 pt. (54%) had fever, typical biliary pain was present in 27 pts. (47%) of them. Symptoms of complete biliary obstruction such as clay coloured stools, high coloured urine were present in 41 pts. (71%). Cachexia was seen in 13 pts. (22%). The liver was palpable in 24 pts. (42%) gall bladder felt in 18 pts. (31%).

The mean serum bilirubin level was 16.41 mg. The range being 2.0-40 mgs. The mean alkaline phosphatase level was 452.95 Iu in the range 102-1132 IU. Urine urobilinogen was absent in 34 (59.65%) patients. Serum albumin was in the range of 3-5 gms. Ultrasound showed Intrahepatic biliary dilatation is 83% of cases. ERCP was done in 21 pts as a diagnostic as well as therapeutic modality for CBD stone retrieval and sphincterotomy and for stenting in patients with biliary strictures. All patients received 3 doses of Vit.K. preoperatively. Preoperative prophylactic antibiotic were

used in all .

In our study we found the main cause of biliary obstruction was due to the choledocholithiasis (18 pts. 31.58%) of these majority were female . The next most common cause was malignancy which included carcinoma head of pancreas and periampullary carcinoma as a group (15pts. 26.32%)

Since the major cause of obstructive Jaundice, was Choledocholithiasis in our study a curative treatment was given to them. Other patients with benign pathologies too had a curative treatment and hence the total amount of patients who had curative treatment was 30 (52.63%). The palliative procedure were done mainly for patients diagnosed to have carcinoma head of pancreas and various components of periampullary carcinoma and they did well in the immediate post operative period. Only 3 patients of carcinoma head of pancreas and periampullary carcinoma group underwent curative pylorus preserving pancreaticoduodenectomy and they did well. A total of 10 patients succumbed to their disease in the course of study who presented mainly with advanced cholangiocarcinoma and carcinoma of gall bladder.

We found a major complication of persistent post operative biliary leak in 3 persons of 57 pts. Operated. Of these 2 patients recovered well and 1 patient lost follow up. One patient with carcinoma from lower CBD is still surviving 20 months after surgery. Patients with benign disease are on regular follow up and doing satisfactorily.

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AGE	NO. OF PATIENTS
<20 years	1 (1.75%)
20-35 years	10 (17.54%)

36-50 years	18 (31.58%)
51-65 years	23 (40.35%)
>65 years	5 (8.77%)
Total	57

AGE DISTRIBUTION

BILIRUBIN LEVELS	NO. OF PATIENTS
<5 mg%	2 (3.51%)
5-10 mg%	19 (33.33%)
11-20 mg%	19 (33.33%)
21-30 mg%	7 (12.28%)
> 31 mg%	10 (17.54%)

BILIRUBIN DISTRIBUTION

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DIAGNOSIS

CARCINOMA HEAD OF PANCREAS	6	2	8(14.04%)
PERIAMPULLARY CARCINOMA	6	1	7(12.28%)
STRICTURE CBD	4	2	6(10.53%)
CARCINOMA GALL BLADDER	3	2	5(8.77%)
CHOLEDOCHAL CYST	0	3	3(5.26%)
CHOLANGIO CARCINOMA	4	1	5(8.77%)
CBD STONES WITH CHOLELITHIASIS	7	11	18(31.58%)
OTHERS	4	1	5(8.77%)

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MANAGEMENT

	34	23	57
CARCINOMA HEAD OF PANCREAS	1	6	1
PERIAMPULLARY CARCINOMA	2	4	1
STRICTURE CBD	4	2	0
CARCINOMA GALL BLADDER	1	1	3
CHOLEDOCHAL CYST	3	0	0
CHOLANGIO CARCINOMA	0	1	4
CBD STONES WITH CHOLELITHIASIS	15	2	1
OTHERS	4	1	0
TOTAL	30(52.63%)	17(29.82%)	10(17.54%)
DISCUSSION			

Gallstones

DISCUSSION

Jaundice is a distressing problem for any human, more so when they are ignorant of the severe underlying pathology. The presentation is usually very late with many patients trying self medications. It is attributable also to the paucity of symptoms. Onset of specific symptoms also implies that the disease is advanced and involvement of vital structures.

Comparing the studies elsewhere too , the observation in our study also shows an increase in incidence in males in general and benign conditions being more common in female. The age of onset of disease is also low including the malignancies. The overall average age of incidence of surgical jaundice is 49 years and the average age for malignant diseases is 52 years. The lowest age recorded for a male patient is 19 years and for a female is 25 years both diagnosed to be with choledocholithiasis .

Comparing with S.Agal et al of Mumbai who studied 62 cases of malignant etiology and M.Kannan et al of Chennai who studied 455 cases of both benign and malignant etiology there is more or less equal age incidence .

In our study liver was palpable in 42% of patients only while in Benjamin series it was palpable in 50% of the patients and in the Popper series is 54%.The gallbladder was palpable in 31% of our patients while in Benjamin series it was palpable in 50% of the icteric patients and 62.20% of those with pancreatic malignancies.

Ultrasound was done in all our patients . It showed dilatation of intrahepatic biliary radicles in 83% of patients. All the choledocholithiasis patients underwent cholecystectomy and choledocholithotomy either by ERCP sphincterotomy and stone retrieval or CBD exploration with T - Tube are doing well.

Of the 15 patients with pancreatic carcinoma ,10 patients underwent bypass procedures and 3 patients underwent Whipples pancreaticoduodenectomy followed by chemotherapy.

Comparing to other studies of Benjamin and Popper , our study showed equal curative rates in the management of other benign extrahepatic biliary tract obstructive lesions such as stricture of the Common Bile Duct , Choledochal cyst and patients

presenting with chronic pancreatic bile duct stricture.

We had 2 patients with Hepatocellular Carcinoma who were referred to higher centres for evaluation and management. Only 1-12 % of HCC patients manifest obstructive jaundice as the initial complaint. Identification of this group of patients is clinically important, because surgical treatment may be beneficial.

In our study , we did not attempt any modality of preoperative biliary drainage for any amount of jaundice mainly in patients with malignant cause of obstructive jaundice since various studies have shown no change in the survival benefits with this procedure. Hence ERCP and stenting was done only for benign cases with features of cholangitis and sepsis who recovered very well.

We had 10 deaths in the follow up and those under investigations. These patients were mainly in their terminal stage of their illness and the underlying pathology was mainly advanced cholangiocarcinoma and advanced carcinoma of the gallbladder. Though the death percentage was a little higher compared to other studies done elsewhere it cannot be considered significant as the study group was small .

CONCLUSION

The following conclusions were made :

- * Obstructive jaundice was more common in men. Benign conditions such as choledocholithiasis was common in female and malignant conditions were common in male.
- * Most of them in the late fifth and sixth decades of life
- * Choledocholithiasis was found to be the most common cause of obstructive jaundice; tumours (pancreatic carcinoma , periampullary carcinoma and cholangiocarcinoma) are next frequent causes.
- * Biliary obstruction due to metastasis is not uncommon.
- * Ultrasound followed by endoscopic retrograde cholangiopancreatography and Computed Tomography are the investigation of choice.

* Patients with benign pathology have a favourable outcome and most of them had improved remarkably and had returned to normal activity.

- Palliative bypass procedures are the final outcome in most malignancies causing biliary obstruction; their use being not in prolonging life span but in giving a better quality of life.

•

PROFORMA

NAME :

AGE:

SEX :

ADDRESS:

UNIT:

O.P/I.P No.:

Socioeconomic status:

D.O.A:

D.O.D.:

PRESENTING COMPLAINTS :

SYMPTOMS :

SIGNS :

GENERAL

Built :

Pallor :

Icterus :

Pedal oedema :

Scratch marks:

Hydration:

Signs of liver failure

Clubbing

BP :

PR:

ABDOMEN

- Jaundice
- Pain
- Malaise
- Loss of wt & appetite
- Itching
- High coloured urine
- Pale stools
- Fever
- Mass
- Others
- Palpable liver
- Palpable gall bladder
- Mass
- Tenderness

Gallstones

- Ascites
- Others

OTHER SYSTEMS :

INVESTIGATIONS

Blood :

Complete haemogram - Hb., TC, DC, ESR

Sugar, Urea

Serum electrolytes - Na, K, Cl, HCO₃

Serum creatinine

Liver function tests - Sr. Bilirubin (total & direct)

Sr. Proteins (A:G. Ratio)

Enzymes (SGOT, SGPT, SAP)

Bleeding & Clotting time, Prothrombin time.

Urine :

Routine - alb, sugar, deposits,

BS, BP, Urobilinogen

Imaging :

X-ray chest P.A. & abdomen A.P.

USG abdomen

CT scan

ERCP

Others

Gallstones

PREOPERATIVE PREPARATION

IV fluids, Vitamin K, Nutrition, Antibiotics

OPERATIVE PROCEDURE

Dt:

Surgeon :

Anaesthetist

Anaesthesia

Procedure

Findings :

Recovery

Post Operative complications / morbidity

Relief of symptoms :

FOLLOW UP :

Gallstones

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Gallstones

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